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The Emperor Has No Causation: Exposing a Judicial Misconstruction of Science

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COMMENTARY

THE EMPEROR HAS NO CAUSATION: EXPOSING A JUDICIAL MISCONSTRUCTION OF SCIENCE

JOHN G. CULHANE*

I. INTRODUCTION

In his novel, *The Information*, Martin Amis describes the relentless and systematically increasing humiliation of the human race throughout the ages.¹ According to Amis, much of this has to do with how science continually reveals us to be less of the Big Cheese of the Universe.² We once thought the sun revolved around us; now we know better. More recently, the discovery of planets outside of our solar system leads us to realize that we are probably not a unique life form³ — further humiliation.

Courts, sometimes of necessity, have had a sense of deep humility — if not embarrassment — about their place in the universe for quite some time; they have long claimed that they do not know enough to recognize a particular cause of action,⁴ or that, even when the action is recognized, there is simply not enough information available to grant recovery.⁵ This judicial modesty has led

1. MARTIN AMIS, THE INFORMATION (1995).

3. See David Chandler, Earth is Not Alone: We Could Have A Sister Planet by 2010, BOSTON GLOBE, Mar. 4, 1996, at 25. The author notes that the Hubble telescope has thus far enabled us only to discover large planets, but, "[i]n as little as 10 to 15 years it may become possible to take one of the most exciting steps . . . [:] capture a picture of an Earth-sized planet orbiting another star, and even analyzing its atmosphere to look for evidence . . . that might indicate the existence of life forms inhabiting its surface." Id.

4. This "modesty" was most pronounced in the emotional distress cases discussed infra Part II.

5. In the torts arena, cases involving time lags between impact and injury typically generate the most problems, especially when the causal link between the two is only dimly understood. Of course, current problems in this area are most apparent in so-called "toxic torts" cases. See discussion infra Part II. But certain more traumatic — and visible — impacts have long given rise to difficulties in ascertaining causation. For example, where the plaintiff alleges that cancer resulted from such an impact, courts have been divided as to whether such a theory should be allowed to reach a jury. Compare Kramer Serv., Inc. v. Wilkins, 186 So. 625 (Miss. 1939) (holding court erred in refusing jury instruction that piece of broken glass could not have caused plaintiff's cancer, as the "exact cause of cancer [is] unknown"), with

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^{2. &}quot;It would seem that the universe is thirty billion light years across and every inch of it would kill us if we went there. This is the position of the universe with regard to human life." *Id.* at 149.

to the denial of relief to injured and deserving victims across a whole range of misconduct. Recently, this disturbing practice has been most evident in toxic torts,⁶ where courts at times have uncritically ingested the party line of defendants, and denied recovery where plaintiffs have "failed" to make causal connections with the level of certainty that would satisfy a court.⁷

In different ways, the comments of both Dr. Jack Snyder and Mr. Allan Kanner further the debate about the problems and promise of causal ascription. Dr. Snyder exhorts us to be clear about the terms being used, and his writings reveal a sharp understanding of the difficulties of causal inference.⁸ Further, many of his suggestions would doubtless aid in the resolution of difficulties that courts now seem to find intractable.⁹ After all, toxic torts do raise specific difficulties in causal attribution that rarely surface in routine cases. Between the lines, however, Dr. Snyder turns out to be less a friend of deserving plaintiffs than I. Throughout the following remarks, I will point to areas of divergence between us, focussing on statements that I believe are open to the same charge of "fuzziness" he levels against his legal side — which at times he seems to regard as an "evil twin."¹⁰

Allan Kanner, on the other hand, in both his writings and his practice, has been a relentless advocate of plaintiffs injured by what he calls "synthetic living"

6. The term "toxic torts" has sometimes (and misleadingly) been used to cover a whole realm of legal responses to the environmental and human costs of toxic exposures. See JEAN MACCHIAROLI EGGEN, TOXIC TORTS IN A NUTSHELL 1 (1995). For purposes of this Article, the term is used to signify a civil lawsuit involving toxic exposure brought under more or less standard and unexceptionable theories of tort law: battery, negligence, strict liability, and nuisance, for example.

7. See discussion infra Part II exploring the causation problems.

8. See generally Dr. Jack W. Snyder et al., Injury and Causation on Trial: The Phenomenon of "Multiple Chemical Sensitivities," 2 WIDENER L. SYMP. J. 97 (1997) [hereinafter Snyder, Multiple Chemical Sensitivities]; Deborah E. Spivack & Jack W. Snyder, Breast Implant Cases, in HANDLING OF SOFT TISSUE INJURY CASES ch. 32 (1993) [hereinafter Spivack & Snyder, Breast Implant Cases]; Jack W. Snyder, Medicolegal Controversies in Proof of Causation and Injury in Occupational Disease and Toxic Tort Cases, in CONSEQUENCES OF TRAUMA: MEDICOLEGAL CONTROVERSIES 365 (1992) [hereinafter Snyder, Medicolegal Controversies].

9. See, e.g., Medicolegal Controversies, supra note 8, at 382-88 (including useful definitions of terms in workers' compensation cases, as well as a schematic diagram suggesting an analysis of "the interaction between environmental agents and biological matrices"); Breast Implant Cases, supra note 8, at 484-95 (reviewing and criticizing various case management techniques).

10. See infra notes 71-75 and accompanying text.

Daly v. Bergstedt, 126 N.W.2d 242, 247 (Minn. 1964) (holding testimony of one doctor, representing a minority view on cancer causation, sufficient to uphold jury verdict in plaintiff's favor where site of impact later developed a cancer). A capable discussion of such cases is found in Bert Black & David E. Lilienfeld, *Epidemiological Proof in Toxic Tort Litigation*, 52 FORDHAM L. REV. 732, 739-44 (1984).

and "technological failure."¹¹ Since I agree with much of what he says, the comments that follow are less a direct critical response to Mr. Kanner, and more a further exploration of his provocative remarks.

This Commentary is structured as follows. Section II begins by offering a general discussion on causation and causal theory. Then I attempt to locate the toxic tort causation controversy within the broader matrix of casual problems that have been making courts squirm at least since the time that a disappointed seeker of ale swung at the ale house owner's wife, and missed.¹² We shall see that, when faced with new "categories" of injuries, courts have moved from clumsy (and later embarrassing) wholesale rejection to eventual recognition. In the end, the fact of injury often proves too compelling for courts to continue to ignore. Yet courts continue to misunderstand causation, with sometimes unfortunate (perhaps indefensible) results.

Next, these points are specifically applied to toxic torts. I explore how the misunderstanding of causation can lead to odd results, for both plaintiffs and defendants. Express use of a more fluid, probabilistic model of causation would aid courts in avoiding some of the difficulties that they now face.

In a brief Part III, I conclude the substantive discussion of this Commentary piece with a consideration of the jury's role in assessing causation as I have recast that concept. I suggest that the question of the blameworthiness of the defendant's conduct is not fully separable from the conclusion on causation that the jury reaches; I then complete the heresy by endorsing this unholy mix of doctrine.

II. PROBLEMS IN CAUSATION

The issues arising in toxic torts only seem new. Careful scholars, including both Dr. Snyder and Allan Kanner, recognize that such problems have existed forever.¹³ Kanner notes that "[1]aw has long known injuries which cannot be determined and which change in nature³¹⁴ Why, then, have courts treated

^{11.} Allan Kanner, The Politics of Toxic Tort Law, 2 WIDENER L. SYMP. J. 163 (1997) [hereinafter Kanner, Politics of Toxic Tort]. See also Allan Kanner, Environmental Justice, Torts and Causation, 34 WASHBURN L.J. 505 (1995) [hereinafter Kanner, Environmental Justice]; Allan Kanner, The Evolving Jurisprudence of Toxic Torts: The Prognosis for Corporations, 12 CARDOZO L. REV. 1265 (1991); Critique of Compensation Systems: Response to Rosenberg, Vol. 1, No. 3, COURTS, HEALTH SCIENCE AND THE LAW, Georgetown University Medical and Law Centers (Jan. 1991).

^{12.} I. de. S. & Wife v. W. de S., 1348 [or 1349] LIBER ASSISARUM, folio 99, PLACITUM 60, reprinted in RICHARD A. EPSTEIN, CASES AND MATERIALS ON TORTS 66 (6th ed. 1995).

^{13.} Symposium, Current Issues in Toxic Torts, 2 WIDENER L. SYMP. J. 1 (1997) [hereinafter Toxic Tort Symposium].

^{14.} Kanner, Politics of Toxic Tort, supra note 11, at 169.

toxic torts cases so gingerly, worrying more about the difficulty of proving causation than they do in other cases?

Dr. Snyder provides a clue to the answer. In one of his writings,¹⁵ he provides the example of an eyewitness account of someone falling on ice. Instinctively, we draw the inference that the ice caused the fall, since we "know" that the presence of ice increases the chance of a fall.¹⁶

What can we say about this knowledge? Why is it more comfortable than the sort of confidence we place in causal conclusions in toxic tort cases? Much of the answer is that both judicial and lay notions of causation are based on an older, now rightly discredited, version of this concept. A brief account of this notion of causation will enable us to see how it continues to fix the stare of the judiciary.

A. The Evolution of Causal Theory

As Dr. Snyder points out, until this century, the governing causal paradigm was mechanistic.¹⁷ One object was thought to exert force on another, thereby bringing about a certain and predictable result.¹⁸ Causation was conceived of as, primarily, a deductive process, whereby so-called "covering laws" were created and then expanded to fit ever-expanding sets of natural phenomena.¹⁹ These covering laws, which were really a set of deductive principles, were then used to explain the previously unexplainable. The whole process was thought to proceed inexorably, leading always to more and more universal knowledge.²⁰

Today, and for most of the present century, the dominant notion of causation has been inferential.²¹ Based on observation of a connection between two phenomena, causal hypotheses are generated to connect the two. However, these hypotheses are always subject to falsification, because new and more sophisticated experiments are used to test the proferred causal theories.²² Indeed, as Dr. Snyder and others have pointed out, it is the very approach to this

19. See Brennan, supra note 17, at 478, citing to D. BOHM, CAUSALITY AND CHANCE IN MODERN PHYSICS 36 (1959).

20. Brennan, supra note 17, at 479 n.52; see also Edward J. Imwinkelried, Evidence Law Visits Jurassic Park: The Far-Reaching Implication of the Daubert Court's Recognition of the Uncertainty of the Scientific Enterprise, 81 IOWA L. REV. 55, 59-60 (1995).

21. Brennan, supra note 17, at 482.

22. This theory of hypothesis falsification is usually credited to Karl Popper. See KENNETH J. ROTHMAN, MODERN EPIDEMIOLOGY 9 (1986).

^{15.} Snyder, Medicolegal Controversies, supra note 8, at 7.

^{16.} *Id*.

^{17.} See Toxic Tort Symposium, supra note 13; see also Troyen A. Brennan, Causal Chains and Statistical Links: The Role of Scientific Uncertainty in Hazardous-Substance Litigation, 73 CORNELL L. REV. 469, 478-83 (1988) (providing a succinct history of causal theory).

^{18.} See MARIO BUNGE, CAUSALITY AND MODERN SCIENCE 108 (3d ed. 1979) (causation was restricted to "whatever produces a change in the velocity of bodies").

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constant uncertainty that separates the older idea of deductive causation from its accepted, inductive replacement.²³ Whereas relentless and inconclusive hypothesis testing confronts uncertainty under the inferential model, the deductive model regarded such uncertainty as curable. The challenge was to make the unexplained phenomenon fit under the constantly expanding covering laws.²⁴

It is important to recognize that, under the inferential model, the best we can do with causation is to express a level of confidence in our judgment that may be higher or lower depending on a number of related factors including: the number of trials that have been done; our belief that those trials have been wellconceived and executed to test the hypotheses for which they argue; the amount of evidence we have gathered; and whether that evidence is reliable and repeatable.²⁵ Thus, causation produces inescapably probabilistic and indefinite results.²⁶

B. Judicial (Mis)Adventures in Causation

Unfortunately, courts have not always appreciated the advances in scientific theory as thoroughly as they have understood the accompanying explosion of scientific knowledge and information. In the mine run of cases, it appears that courts sometimes cling to the mechanistic view of causation.²⁷ Indeed, the "but for" test that still enjoys currency in causal determinations seems to betray adherence to the earlier view; A either caused B, or it did not.²⁸ And, as

^{23.} Dr. Jack W. Snyder, Injury and Causation on Trial: The Phenomenon of "Multiple Chemical Sensitivities," Address at the *Widener Law Symposium Journal* Toxic Tort Symposium (Mar. 15, 1996), *in* 2 WIDENER L. SYMP. J. 97 (1997); *see also* Margaret G. Farrell, Daubert v. Merrell Dow Pharmaceuticals, Inc.: *Epistemology and Legal Process*, 15 CARDOZO L. REV. 2183, 2193-94 (1994) (explaining differences between deductive and inductive models of causation, and emphasizing the insight of the inductive model that it is impossible to predict the future with certainty).

^{24.} See Farrell, supra note 23, at 2193; Brennan, supra note 17, at 479 n.52.

^{25.} ROTHMAN, supra note 22, at 4.

^{26.} See Imwinkelried, supra note 20, at 60-63 (applying this insight scientific enterprise more generally).

^{27.} Glen O. Robinson, Probabilistic Causation and Compensation for Tortious Risk, 14 J. LEGAL STUD. 779, 780 (1985) ("[0]pinions still largely reflect the dominant mechanistic and deterministic conceptions of causation[,]" which "may do no great harm in the routine accident case."). See Stimpson v. Wellington Serv. Corp., 246 N.E.2d 801 (Mass. 1969), discussed infra note 32.

^{28.} As a purely evidentiary matter, this observation oversimplifies matters; the test is really whether one can say that, more likely than not, B would not have occurred in the absence of A. The reasoning of the jury in these cases is likely probabilistic to an extent. But the point here is that the statement of the legal rule assumes a binary choice.

Professor Rosenberg²⁹ has pointed out, this approach usually causes no great harm.³⁰ When A strikes B, who then begins immediately to bleed, or — to return to our earlier example — when B slips on an icy sidewalk that A failed to shovel, the choice between inductive and deductive reasoning makes no practical difference. Because we have so many examples of the causal connection, and because evidence for it is so easily obtained (through visual observation), the probability of association is so great, that it is easy, and usually harmless, to substitute a mechanistic view of causation for the strong inductive inference that we are warranted in drawing.³¹ Indeed, the mechanistic view can even help the plaintiff in certain cases, by assuming a connection that would otherwise have to be shown by probabilistic evidence.³²

But this same reliance on mechanistic notions of causation can spell trouble for courts in cases where the connection is not so apparent. Toxic torts, of course, supply a dramatic example of this problem because toxins often invade the body surreptiously, and cause harm only after a long period of time.³³ However, toxic torts is not the only area in which courts have notoriously had a difficult time in deciding whether or not to grant recovery; emotional distress claims are another set of cases that have presented a problem.

1. Emotional Distress Claims

The myriad of problems accompanying emotional distress claims have repeatedly been stated by courts, and thus, are well known to most of us. These problems include: the difficulty of separating the meritorious claims from the feigned ones; the resultant "flood of litigation" that would be undammed were

^{29.} Professor Rosenberg is a law professor at Harvard University who represented plaintiffs in DES cases, including Payton v. Abbott Lab., 512 F. Supp. 1031 (D. Mass. 1981); Sindell v. Abbott Lab., 607 P.2d 924 (Cal. 1980).

^{30.} David Rosenberg, The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System, 97 HARV. L. REV. 851, 855 (1984).

^{31.} The beguiling simplicity of such examples is questioned *infra* note 58 and accompanying text.

^{32.} See Stimpson, 246 N.E.2d at 805. There, adherence to a mechanistic view of causation helped the plaintiff by relieving him of the burden of showing that the force of defendants' truck exerted on the pipes beneath the street in turn caused the pipes in plaintiff's basement to burst. *Id.*

^{33.} See Robinson, supra note 27, at 779 (on the "lag time" between exposure and injury). Environmental pollutants, such as PCB's and dioxin, are the types of toxins that often enter the human organism without being noticed. *Id.* Other toxins, such as those contained in pharmaceutical products, may also be unnoticed in the sense that the consumer is aware that she is taking a certain drug, but often is *not* aware that the drug poses a particular toxic risk.

the claims to be recognized; and the remoteness of the harm from the defendant's perspective in unleashing the risks he did.³⁴

Yet all of these concerns can, without undue strain, be traced back to a fundamental sense that causation, defined mechanistically, is too difficult to establish. Sometimes, as in *Mitchell v. Rochester Railwary Co.*,³⁵ the court's language comes close to expressly revealing this concern, which is usually camouflaged. In *Mitchell*, the court stated the "difficulty . . . in determining whether [injury] exist[s], and if so, whether [it] was caused by the negligent act of the defendant, would . . . be greatly increased" where the claim involved a "scare" which resulted in physical and mental suffering.³⁶ It might have been even more honest for the court to have said that it simply did not believe, despite the medical evidence adduced at trial, which, incidentally, the jury resolved in plaintiff's favor, that the fright caused the physical injuries of which the plaintiff complained. In *Mitchell*, the resultant injuries were quite severe; the plaintiff suffered a miscarriage.³⁷ Apparently, even an injury so patently physical does not fit within a causal paradigm that demands immediacy of result from impact.

Mitchell was decided just before the turn of the century, and the tide turned in plaintiffs' favor shortly thereafter.³⁸ But continued adherence to a mechanistic model of causation has proven stubborn. In fact, the impact rule instituted by courts when recovery for negligently inflicted emotional distress began to be recognized is also quite mechanistic in inspiration. The powerful symbolism of an impact — a "hit" — gave courts false but durable comfort that the injuries that followed were genuine, so that such an impact was required for recovery.³⁹ Even today, the majority of courts require at least some physical injury

39. See, e.g., Kenney v. Wong Len, 128 A. 343, 344 (N.H. 1925) (allowing plaintiff to recover when a mouse hair in stew touched her mouth); Porter v. Delaware, L. & W. Ry., 63 A. 860 (N.J. 1906) (permitting recovery when the plaintiff got dust in her eyes). That these impacts were slight only reinforces the point that the reassurance they provided courts was false.

^{34.} See RICHARD A. EPSTEIN, CASES AND MATERIALS ON TORTS 544 (6th ed. 1995); W. PAGE KEETON ET AL., PROSSER AND KEETON ON THE LAW OF TORTS 360-61 (5th ed. 1984) (also expressing concerns in compensating harm that is "often temporary and relatively trivial" and the possible over-deterrence of merely negligent defendants).

^{35. 45} N.E. 354 (N.Y. 1896).

^{36.} Id. at 355.

^{37.} Id. at 354.

^{38.} Consequently, in Battalla v. State, 176 N.E.2d 729 (1961), the New York Court of Appeals overruled the *Mitchell* decision. "It is undisputed that a rigorous application of its rule would be unjust, as well as opposed to experience and logic." *Id.* at 730; *see also* Dulieu v. White & Sons, [1901] 2 K.B. 669, 682. The English court recognized the legitimacy of claims based "solely" upon emotional distress, and dismissed arguments about remoteness of damage and untrammeled litigation. *Id.*

accompany the mental suffering in order for a plaintiff to recover.⁴⁰ Obviously, courts have a lingering disquiet with what they cannot see, or what cannot be fully explained mechanistically.⁴¹

2. Toxic Torts

In toxic torts, the courts' dogged insistence, albeit often tacit, on a mechanistic kind of causation has created no end of problems, and for both plaintiffs and defendants. The most easily identified effect of this world-view harms defendants. In a number of cases, courts have given bizarre and undue deference to talismanic statements by treating physicians that the plaintiffs' injuries were caused by exposure to the (allegedly) toxic substance in question. In Sterling v. Velsicol,⁴² for example, the court announced that recovery could be granted where a physician testified to the causal connection "to a reasonable medical certainty."43 In fairness, the court stated that it was the underlying science, not the terminology, that was important.44 Yet a reading of the Velsicol decision unmistakably leaves one believing the court felt more comfortable with the mechanical model of causation, even where the mechanism itself was not understood. For example, the Velsicol court referred approvingly to the expert testimony of physicians who said both that "no one knows what causes cancer" and - paradoxically - that the defendant's chemicals had caused the cancer "based upon a reasonable medical certainty."45

Of course, in these cases the plaintiffs also present general evidence of the substance's ability to injure, the plaintiff's exposure to the substance, and even supporting evidence of the treating doctor herself. But the court's heavy reliance

42. 855 F.2d 1188 (6th Cir. 1988).

43. Id. at 1200.

44. Id. "[T]his standard implicates the qualifications of the witnesses testifying, the acceptance in the scientific community of their theories, and the degree of certainty as to their conclusions." Id.

45. Id. at 1203; see also Cantrell v. GAF Corp., 999 F.2d 1007, 1012-13 (6th Cir. 1993) (upholding doctor's testimony that asbestos had caused the plaintiff's cancer of the larynx).

^{40.} See, e.g., Dziokonski v. Babineau, 380 N.E.2d 1295, 1302 (Mass. 1978); 3 FOWLER V. HARPER ET AL., THE LAW OF TORTS § 18.4, at 696-97, 679 n.41 (2d ed. 1986).

^{41.} That is not to say that courts do not have other reasons for denying recovery in these cases. In one of the most indefensible decisions in recent memory, the Texas Supreme Court, in Boyles v. Kerr, 855 S.W.2d 593 (Tex. 1993), declined to recognize a cause of action for negligent infliction of emotional distress on behalf of a young woman who had been covertly videotaped having sex, even though the tape was shown to at least ten people, and thereafter, "discussed" by many others, sometimes in plaintiff's presence. *Id.* at 595-96. The court seemed to imply that physical manifestations of injury would have changed the result, but its decision is less about proof — because no one would deny that the plaintiff in fact suffered serious emotional distress — and more about a misguided concern with litigation run rampant.

on a simple statement by the physician betrays a "humiliating humility." Since a court cannot find the mechanistic connection (subcellular damage is, by definition, much smaller than an icy sidewalk), it may "panic" and cede responsibility for making this kind of connection to the physician. This is neither good science nor good law. As we will see, however, there is a place for statements of treating physicians whose special familiarity with the patient's history may suggest causal connections that might otherwise be missed.⁴⁶ This mechanistic view can also be injurious to plaintiffs, but seeing how that is so is a bit more elusive. Enthralled by the mechanistic model, courts, as in Velsicol have constructed an overly rigid causation structure that misses the dynamic interconnection between different kinds of proof. In toxic exposure cases, courts generally require that plaintiffs make several independent showings. The first of these is that the substance in question could have caused the injury of which plaintiff complains.⁴⁷ This usually means that the plaintiff must come forward with some kind of epidemiological evidence linking exposure to the increased risk of contracting whatever illness the plaintiff is alleged to have suffered.48 However, sometimes the strength of association between exposure and illness is so strong that plaintiffs are excused from proof at this level. Thus, for example, the risk of developing asbestosis and mesothelioma from asbestos exposure is so well documented that plaintiffs with the appropriate injury have no difficulty with causation at this stage of their case.49 If such strong epidemiological evidence is weaker, or unavailable, some courts have allowed plaintiffs to proceed based on biological plausibility, as supported by animal tests.

If this showing of generic causation is made, plaintiffs can then proceed to show that they were, in fact, exposed to the alleged toxin. This component is largely temporal, focusing on the sequence by which a victim comes to be assaulted by a particular toxin. At times, it is the very question of *any* exposure that is at issue. It might be clear enough (for purposes of litigation) that the challenged substance can be toxic, but the plaintiff might have been at a distance, thus making exposure questionable. If the substance is not one that leaves physiological souvenirs,⁵⁰ this question can assume central importance. More

49. EPSTEIN, supra note 34, at 843.

50. Sometimes these traces are readily discernible. *See In re* Paoli R.R. Yard PCB Litig., 35 F.3d at 756 (doctor tested each plaintiff for PCBs); Gideon v. Johns-Manville Sales Corp.,

^{46.} See infra notes 65-70 and accompanying text, discussing In re Paoli R.R. Yard PCB Litig., 35 F.3d 717 (3d Cir. 1994), cert. denied, 115 S. Ct. 1253 (1995).

^{47.} See Velsicol, 855 F.2d at 119; see also Ilhardt v. A.O. Smith Corp., 168 F.R.D. 613, 620 (S.D. Ohio 1996) (stating generic causation means "that it is merely possible for the identified harm to occur").

^{48.} Epidemiology is the science that attempts to quantify "the occurrence of illness." ROTHMAN, *supra* note 22, at 23. Epidemiologists try to determine whether causal associations are present between exposures and diseases that follow. *Id.*

typically, the exposure question is whether the plaintiff's exposure was sufficient to have caused the injury suffered. This issue was squarely addressed by Judge Weinstein in the well-known asbestos class action litigation, *In re Agent Orange Product Liability Litigation*,⁵¹ in which he observed that the level of exposure plaintiffs claimed placed them outside of the category of persons that had been epidemiologically linked to disease.⁵² Thus, the exposure question can surface as a component of the generic causal inquiry. Likewise, exposure issues can glide easily into the question of individual causation. For example, if the plaintiff can demonstrate sufficient exposure to have resulted in the complained injury, it may be a short step to the conclusion that the injury did *in fact* result from that same exposure. Yet the questions are analytically distinct.

The final step in the causal analysis, for plaintiffs who get this far, is to show that *their injury* was caused by the toxin. This so-called "specific causation" requirement is needed because epidemiology can only supply group information — members of the exposed group were more likely than others in the population to develop the illness or injury in question.⁵³ Similarly, laboratory studies can show biological links, but this is not the same as demonstrating individual causation, largely because of the unique environmental circumstances of each injured person.⁵⁴ These differences may arise both in the external environment, where the interaction of other ambient substances blurs the role of the toxin, and in the body itself — a result of the differences in physiology and genetic makeup among members of the human species.⁵⁵ Since recovery in tort

51. 611 F. Supp. 1223, 1247 (E.D.N.Y. 1985), aff'd, 818 F.2d 187 (1987).

52. Id.

53. This assumption has been articulated and defended by a virtually unanimous voice of courts and commentators. *See, e.g., Velsicol*, 855 F.2d at 1200; Robinson, *supra* note 27, at 855-58.

54. This problem has been particularly acute in the cases of asbestos-exposed plaintiffs who were also smokers. Since both of these exposures significantly increase the risk of lung cancer, isolating the asbestos exposure as *the* cause of the injury is especially challenging. *See*, *e.g.*, Borman v. Raymark Indus., 960 F.2d 327 (3d Cir. 1992). There, the court's extensive analysis of the applicable Pennsylvania law regarding the apportionment of damages between cigarette smoking and exposure to asbestos assumed a contribution by each of the two. The problem, of course, is in figuring out the actual percentage of contribution by each in any one case. The court was ultimately unwilling to assign percentages. *Id.* at 335.

55. Not everyone exposed to a particular toxin will develop the most serious possible diseases; some may not experience any problems from that exposure. For example, lung cancer results from smoking in far greater numbers than in the general population, but the disease is not limited to smokers. Genetics is thought to partially explain why some smokers get lung cancer and some do not. For a comprehensive scientific discussion of the interplay between smoking, lung cancer, and genetic factors, see Tariq Sethi, *Lung Cancer: Science*,

⁷⁶¹ F.2d 1129, 1137 (5th Cir. 1985) (plaintiff was exposed to asbestos). Other injuries are subclinical, so that the dual inferences of injury and exposure are even more difficult to draw. *Id.*

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depends critically on making the individualized connection between defendant's wrongful conduct and plaintiff's injury, this final step is seen as indispensable.

This terraced approach to the question of causation betrays an allegiance to the mechanistic model of causation. By requiring hard proof of a certain level of exposure as well as a showing of individual causation, courts depreciate the fact that *all* causal proof — generic, exposure-related, and specific — is probabilistic.⁵⁶

As a corollary observation, at least where the epidemiology supports a causal connection, judicial familiarity with medical testimony and *sub rasa* adherence to mechanistic thinking means that courts and juries "can't wait" to get to the individualized proof.⁵⁷ Thus, *this* plaintiff's cancer was caused by *this* defendant's toxin — in much the same way the icy sidewalk caused the fall. The doctor said so.

Well, what's wrong with that? Tort law — all law, in fact — rightly insists on a showing of individual causation, because otherwise, as Dr. Snyder points out, we are imposing liability for the creation of risk. Dr. Snyder and I are in agreement that liability in tort should continue to require proof of causation, not simply that the defendant created an unreasonable risk; otherwise, society would be better served by switching to some type of compensation scheme.

However, the problem with the step-by-step approach adopted by courts is that, in failing to appreciate that all causal determinations are inescapably probabilistic, courts miss the subtle interactions between the categories of causation created by this step-by-step approach. It might be, for example, as with the link between asbestos and asbestosis, that the epidemiological associations are sufficiently strong that a reasonable inference of causation is warranted without a further, particularized showing. Courts recognize this point only in clear cases like asbestosis,⁵⁸ however, they miss the implications of

Medicine, and the Future, 314 BRITISH MED. J. 652 (1997).

56. Professor Rosenberg summed this point up eloquently: "'Particularistic' evidence ... is ... no less probabilistic than is the statistical evidence that courts purport to shun. All knowledge of past as well as future events is probabilistic." Rosenberg, *supra* note 30, at 870.

57. A rare glimpse into judicial discomfort with epidemiological proof is found in JONATHAN HARR, A CIVIL ACTION 237-38 (1995). Relating the details of a pre-trial conference discussion between the judge assigned to the case, Judge Skinner, and the plaintiff's lawyer, Jan Schlichtmann, Jonathan Harr relates the following:

[Judge Skinner]: "Well, specifically, are you going to use the -"

"I will not use statistics to prove causation," said Schlichtmann.

[Judge Skinner]: "Okay, fine. Then my problems for the time being are resolved."

58. The DES cases, well-known for the creation of the novel market share liability concept, provide an example, alongside asbestos, of a situation in which the product is

this insight in cases where the inference is less powerful. Why doesn't a strong causal association, supported by powerful epidemiological studies, supply at least some evidence that any particular plaintiff's injury was caused by the defendant's toxin? And, why can't a careful medical examination, supported by available science, suffice for recovery in cases in which the epidemiology either has not been done, or is equivocal, as is often the case?

What should courts do in order to respect the interaction of the various probabilities that should rightfully figure into a decision on the causation issue? The stubborn difficulty of these cases calls for two responses, neither of which is especially surprising, much less radical.

First, courts should continue to perform the gate-keeping function they have always served.⁵⁹ Even Professor Nesson, who served as an expert witness for the plaintiffs in the Woburn, Massachusetts, water contamination case,⁶⁰ concedes that there are cases in which the evidence of *non-causation* is so clear that the case should be dismissed. If, according to Nesson's memorable example, a plaintiff alleged that eating carrots had caused her cancer, the court would be justified in dismissing the claim for lack of evidence.⁶¹ No epidemiological studies support the conclusion, and no science at any level, not even *in vitro* studies or animal studies, have pointed to such an association. To recognize such a claim would be to succumb to the fallacy of *post hoc*, *ergo propter hoc*.

Second, courts should exhibit greater flexibility in allowing plaintiffs' attorneys to range between the different levels of causation on which these cases operate. There is some evidence of this tendency in recent decisions. For example, in *Christophersen v. Allied-Signal Corp.*,⁶² the appellate court overturned the trial judge's grant of summary judgment, noting that the plaintiff's expert had presented enough evidence to reach the jury on the issue whether exposure to defendant's nickel and/or cadmium fumes had caused the plaintiff's cancer. In *Christophersen*, the expert⁶³ considered as pieces of one causal puzzle such diverse questions as dose-response, general chemical and biological knowledge on the carcinogenicity of these elements, and inferences regarding the plaintiff's exposure.⁶⁴

59. See Daubert v. Merrell Dow Pharms. Inc., 113 S. Ct. 2786 (1993).

60. See generally HARR, supra note 57.

61. Charles Nesson, Agent Orange Meets the Blue Bus: Factfinding at the Frontier of Knowledge, 66 B.U. L. REV. 521 (1986).

62. 902 F.2d 362 (5th Cir. 1990).

known, to something approaching an "epidemiological certainty," to cause certain diseases. As far back as 1980, the California Supreme Court casually remarked on the uncontroversial view that "DES . . . causes adenosis" as well as "cancerous vaginal and cervical growths." Sindell v. Abbott Lab., 607 P.2d 924, 925 (Cal. 1980).

^{63.} In this case, the expert did not have the benefit of epidemiological studies. Id.

^{64.} Unfortunately, the summary judgment ruling was reinstated in a subsequent rehearing *en banc*: Christophersen v. Allied-Signal Corp., 939 F.2d 1106 (5th Cir. 1991). The

The Third Circuit Court took the same promising approach in the exhaustive In re Paoli R.R. Yard PCB Litigation,⁶⁵ where the court allowed precisely the sort of synergistic consideration of general and specific causal connections for which I have been arguing. The court displayed an impressive level of sophistication in applying the standard for admissibility set forth in *Daubert* to its consideration of the district court's findings. Thus, the court allowed the plaintiffs' experts differential diagnoses to support findings of specific causation, but only where the physicians had at least consulted the medical records of the plaintiffs on whose behalf they testified.⁶⁶ The court also recognized that, in theory, examination of individual plaintiffs could only support a causal hypothesis to that particular plaintiff.⁶⁷ However, the court effectively qualified that conclusion in important respects by allowing the physicians' more general knowledge of the toxicity of PCB's to form part of the basis of their conclusions.⁶⁸

It is further noteworthy that the court of appeals found the district court's decision too rigorous in rejecting animal studies which suggested a possible causal connection.⁶⁹ Such epidemiological studies that were performed failed to *conclusively* establish a link between PCB's and various diseases. However, the court observed that such failure was quite a different matter from producing studies tending to prove that *no* association existed.⁷⁰ Thus, the animal studies could provide a useful starting place for the physicians, long-time experts on PCB's, from which the differential diagnoses could then proceed.

This approach recognizes the interplay between the different kinds of evidence that establish the probability that defendant's conduct caused plaintiff's injury. Dr. Snyder seems to recognize the appropriateness of proceeding as my comments suggests. In his *Medicolegal Controversies* article, he tempers his distaste for clinical ecology, which seems able to ascribe causal connections without the benefit of any evidence, with solid recommendations for ways in which environmental irritants could be found to have caused a particular person's allergic reactions.⁷¹ The checklist Dr. Snyder proposes

basis of this decision, however, was that the expert testimony lacked the necessary criteria for admissibility set forth in Frye v. United States, 293 F. 1013 (D.C. Cir. 1923). That test, however, has been discarded by the Supreme Court in the recent case of Daubert v. Merrell Dow Pharms. Inc., 113 S. Ct. 2786 (1993). Under the more liberal *Daubert* standard, which requires only that the evidence be "reliable" — not that it be "generally accepted" — the expert testimony in *Christophersen* would likely have been sufficient to reach the jury. *Id.* at 2796.

65. 35 F.3d 717 (3d Cir. 1994).

66. Id. at 752-65.

67. Id. at 782-84.

68. Id. at 784-85.

69. Id. at 779-81.

70. Id.

71. Snyder, Medicolegal Controversies, supra note 8, at 378-82.

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could be used by both plaintiffs and defendants⁷² in making the kinds of sophisticated, probability-based judgments about causation that are needed. Furthermore, his checklist includes items that run along the continuum from what would usually be considered "generic" to "specific" causation.⁷³

Similarly, in his article, Breast Implant Cases, Dr. Snyder has acknowledged that the epidemiology has lagged behind the litigation, so that the causal inferences that plaintiffs seek to draw about systemic problems, such as connective tissue disease, must either come from clinical observation and whatever sketchy science exists, or not at all.⁷⁴ Reading between the lines, Dr. Snyder's view appears to be that such inferences should not be drawn.⁷⁵ But his "checklist" approach represents the better view. Courts have not, in other cases, neatly separated the causal showings into categories of "generic" or "specific" causation, opting instead for an integrated approach. A return to our woeful, skidding plaintiff example will illustrate the point.

3. "Ice" ing on the Cake⁷⁶

Thus, one *could* reanalyze the slippery sidewalk case as involving demonstrations of both generic and specific causation. First, we could ask whether contact with ice *could* cause a person to slip. This question could be answered with epidemiological data on the increased incidence of slipping upon exposure to ice or with physical evidence about the effect of the contact between the molecular structures of ice and different types of footwear. Under this formally separated method of analysis, the next (unrelated) question is whether *this* injured person had, in fact, come into contact with the ice, and then whether the fall was, in fact, attributable to that contact.

Careful examination of this purposely absurd analysis highlights its analytical deficiency. The final question raised, whether the ice caused the fall in question, would usually be answered, as noted above, by simple observation. But this observation only supports the conclusion for which it is offered because there already exists some basic *generic* causal understanding that permits the connection to be made. To consider why this is so, imagine instead that the plaintiff falls on a sidewalk that presents no apparent hazard. Stripped of the "obvious" general connection between ice and falls, the reason

76. "I can't wait . . . to put the icing on the cake." THE B-52'S, Cake, on MESOPOTAMIA (Warner Brothers/Reprise Records 1982).

^{72.} Likewise, such a checklist could be equally useful to the courts.

^{73.} See Snyder, Medicolegal Controversies, supra note 8, at 378-79.

^{74.} Spivack & Snyder, Breast Implant Cases, supra note 8, at 444-45.

^{75.} My conclusion is reinforced by a comment Dr. Snyder made during a short colloquium that followed the major presentations at the symposium; he referred to the causal case put on during some litigations as "a fraud." *Toxic Tort Symposium*, *supra* note 13.

why the plaintiff fell is not as easy to discern, both on the generic *and* on the specific levels of causation. However, a number of observations of people falling under similar circumstances could lead to a general hypothesis concerning a causal connection.

For example, if our observations were that the great majority of those who fell were obviously older people, we might identify age as a risk factor for falling. Such a tentative hypothesis would then be tested in two ways: on the basic level of observation, by acquiring more examples in order to increase the power of this "study"; and on a deeper level, by assaying an investigation of what it is about age that might make one more likely than another to fall. Moving from observation to analysis and further study is the very essence of epidemiology, so that the point about the dialectical relationship between generic and specific causation is reinforced. My conclusion, then, is that the judicial wall between generic and specific causation should be further breached, as it artificially muffles dialogue between them.

It is possible, however, to over-exaggerate this insight. The separation of causation into categories is useful, and should in fact be retained, if only for purposes of emphasis. Just as focusing on different types of causation can show why the "ice" cases are so easy, where other cases involving slips are not, so too can judicial concentration on particular causal questions highlight issues that may be more or less problematic in a given area. In a small number of potential suits,⁷⁷ the case on any one (or more) aspect of causation will be so unprovable that dismissal should properly result. Otherwise stated, the importance of observation should not mean that a single occurrence, without more, suffices for a finding that any particular substance *either* has the ability to injure *or* that it injured the plaintiff in the way alleged. Such a conclusion would be unwarranted either as epidemiology, laboratory science, or plain observation.⁷⁸

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^{77.} The set of such cases will be small because weak or missing inferences of causation will usually impede the injury in question from resulting in a suit. Thus, a significant number of injuries go uncompensated by those who caused them. This point has been insufficiently appreciated, in my view.

^{78.} Consideration of the exposure issue amplifies this point. Absent proof of exposure, imposing liability against the producer of an alleged toxin is incoherent, meaning, no generic causation, no exposure, no specific causation. Imposing liability in such a case would not even be justifiable under a creation of risk analysis, because (by hypothesis) risk has been neither assessed nor established. If the point in the text is read to include "exposure" under "occurrence," the result is still the same in that no attribution of causal connection should be made based on a single exposure, at least where there is no supporting science. Again, there has not yet been any finding of risk.

4. Daubert and Other Things

In short, law should be consistent with science, but one needs to keep in mind the limits of scientific knowledge which, as noted previously, is uncertain and continually evolving.⁷⁹ Therefore, not every claim should be allowed to proceed on the merits, but most should. The Supreme Court has perhaps moved in this direction recently in Daubert v. Merrell Dow Pharmaceuticals, Inc.,⁸⁰ noting that the court should act as a "gatekeeper" to determine whether the challenged evidence is reliable, which, in the case of toxic torts, translates into the question whether it is scientifically valid.⁸¹ Given Justice Blackmun's astute recognition in *Daubert* that "there are no certainties in science,"82 one might argue that plausible doubts about scientific validity should be resolved in plaintiff's favor, at least allowing the full story of causation to reach the jury. This is premised on the fact that science will always be evolving, under the probabilistic, hypothesis-testing model of causation. Although subsequent developments and expansions of scientific knowledge may call into question the wisdom of a decision in favor of *either* side, this kind of falsification has always been a possible result in every case. In fact, the high stakes in toxic tort litigation may make this context a particularly useful crucible for advancing the connections between sketchy epidemiological evidence and biological science on the one hand, and clinical diagnosis, on the other, that otherwise might avoid detection. Allan Kanner is most likely in agreement with this assertion; his paper submitted for this symposium states that a trial has been a success if it has "resolve[d] honest disputes, [its purpose is] not to find universal truths."83

Seen in light of this emerging recognition of the contingent and limited nature of causal ascription, the flurry of intricate and absorbing proposals for dealing with the evidence provided by epidemiology can be misguided. Professor Richard Delgado, for example, proposes that plaintiffs unable to prove "specific" causation (notice that rigid split again) should be permitted to recover according to the "proportion of culpably injured to nonculpably injured members of the class" based upon what the epidemiology shows.⁸⁴ Professor Daniel Farber, on the other hand, opts for a "most likely victim" approach, under which proportional recovery is scrapped in favor of an all-or-

^{79.} See supra notes 2, 3 and accompanying text.

^{80.} Daubert v. Merrell Dow Pharms. Inc., 113 S. Ct. 2786, 2786 (1993).

^{81.} As Justice Blackmun recognized in *Daubert*, testimony that can be characterized as "scientific knowledge" should be admissible. *Id.* at 2795. Knowledge deserves the adjective "scientific," Blackmun continued, if "supported by appropriate validation" methodology. *Id.*

^{82.} Id. at 2795.

^{83.} Kanner, Politics of Toxic Tort, supra note 11, at 180.

^{84.} Richard Delgado, Beyond Sindell: Relaxation of Cause-in-Fact Rules for Indeterminate Plaintiffs, 70 CAL. L. REV. 881, 904 (1982).

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nothing approach. "[T]hose plaintiffs whose injuries were least likely to have been caused by the defendant receive nothing, while those with the highest causation probabilities get full compensation."⁸⁵

At least Professor Farber's approach has the virtue of recognizing that not all of those exposed to a particular toxin are at equal risk. Nonetheless, these and similar proposals are the misguided children of frustration. They over rely on epidemiology which, by its own admission, is an incomplete tool for gauging causal connection in their attempt to find a category-wide solution to the problem. Better to accept the limits of knowledge, and leave determinations of causation in the hard cases where they belong — in the courtroom.

III. THE JURY'S ROLE IN "THE SPECIAL MORALITY OF TORT LAW"86

I would like to turn, finally, to an issue that has been on Mr. Kanner's mind; the role of the jury in these toxic exposure cases.⁸⁷ Where the regulatory regime — the elite discussed, but equivocally by Professor Kanner⁸⁸ — fails to prevent injury, we properly seek the jury's guidance on whether to impose liability on a particular defendant. Here, I would like to advance the doctrinally heretical suggestion that the jury's determinations on causation will be affected by its sense of whether, and to what extent, the defendant has engaged in wrongful conduct. Thus, where defendant's conduct is intentionally wrongful, as where it intentionally dumps known toxins into the water supply, a jury is likely to resolve doubts on causation in plaintiff's favor. As we move down the scale, first pausing at the kind of wrongful risk creation that is the hallmark of negligence law, and then at cases where the defendant makes a strong demonstration that reasonable steps at averting injury were undertaken, factual determinations on causation in the close cases may less often result in plaintiff's favor.

This observation is "doctrinally heretical" because it assumes seepage through the drywall separating wrongdoing and causation. But note, that I am not suggesting that either of these categories can be dispensed with, nor that they do not retain mutually independent significance. Rather, I take a neolegal realist position on this question, borrowing heavily from the seminal work of Wex Malone.⁸⁹ In writing about factual causation some forty years

^{85.} Daniel A. Farber, Toxic Causation, 71 MINN. L. REV. 1219, 1221 (1987).

^{86.} This phrase may have been coined by Ernest J. Weinrib, in his article entitled (not surprisingly), "The Special Morality of Tort Law." Ernest J. Weinrib, *The Special Morality of Tort Law*, 34 MCGILL L.J. 403 (1989).

^{87.} See Kanner, Environmental Justice, supra note 11, at 507-09 (describing trials as "morality plays").

^{88.} See Kanner, Politics of Toxic Tort, supra note 11, at 176-78.

^{89.} Wex S. Malone, *Ruminations on Cause-in-Fact*, 9 STAN. L. REV. 60 (1956). Malone's article represented the next stage in the development of causation analysis initiated by Leon

ago, Malone was the first to recognize that conclusions on factual causation, no less than those on so-called legal causation, are inescapably intertwined with the decision-maker's opinion of whether the defendant's actions were culpable. He stated, "policy may often be a factor when the issue of cause-in-fact is presented sharply for decision."90 Malone noted that a judge may shape the causal inquiry, and that a jury's subsequent determinations on causation may also be affected, by considering the degree of risk to which the defendant subjected the plaintiff.⁹¹ The riskiness of the defendant's conduct, in turn, speaks directly to the wrongdoing that tort law requires. Negligent action can be described as that which imposes an unreasonable risk on another,⁹² while categories of strict liability involve a wholesale determination that an entire type of conduct is so risky that those who engage in it do so at their peril.93 Thus, those who violate the law's proscription against the creation of unreasonable risk can expect such wrongful conduct to silently strengthen the case on causation. Both judges and juries reinforce this result, finding power in one area where mystery and uncertainty abide in another.

As far as determining the extent to which the defendant should be liable for risk-creating conduct, one important focus is on communication and representation.⁹⁴ Thus, if a manufacturer sold a breast implant accompanied with a proper warning about a certain possible problem, such as leaking, and that problem then surfaced, a fact-finder might conclude that the defendant's conduct was not culpable. Compare, of course, the spiraling horror of the cigarette-marketing machine, as to which facts are being uncovered almost each

93. See generally EPSTEIN, supra note 34.

94. These issues are emphasized in my own writings on product liability and educational malpractice. See John G. Culhane, The Limits of Product Liability Reform Within a Consumer Expectation Model: A Comparison of Approaches Taken by the United States and the European Union, 19 HASTINGS INT'L & COMP. L. REV. 1 (1995); John G. Culhane, Reinvigorating Educational Malpractice Claims: A Representational Focus, 67 WASH. L. REV. 349 (1992).

Green, perhaps the prototypical legal realist. In "Rationale of Proximate Cause," published in 1927, Professor Green began the deconstruction of causation by surgically separating causein-fact and legal, or proximate, cause. LEON GREEN, RATIONALE OF PROXIMATE CAUSE (1927). The latter, he noted, was policy-driven and not really about causation at all, but about drawing practical limitations on recovery. *Id.* at 132. Left alone as the only "true" causation, then, factual causation (or cause-in-fact) was supposed to remain free of the policy considerations that drive legal causation analysis.

Professor Richard Wright is a contemporary torch-bearer for the theory that cause-infact can be determined in a policy-neutral way. See Richard W. Wright, Causation, Responsibility, Risk, Probability, Naked Statistics, and Proof: Pruning the Bramble Bush by Clarifying the Concepts, 73 IOWA L. REV. 1001 (1988).

^{90.} Malone, supra note 89, at 61.

^{91.} Id. at 26.

^{92.} See KEETON ET AL., supra note 34, at 360-61.

day proving that the manufacturers knew, but deliberately withheld, information that — we could laconically understate — might have been material to a potential or current smoker.⁹⁵

This emphasis on communication and the plaintiff's fair chance to avoid injury, illustrates a potential problem with Dr. Snyder's comments. Both in his breast implant writing and in his article on *Medicolegal Controversies*, he notes that liability is increasingly imposed for the creation of risk.⁹⁶ First, I disagree with the conclusion. Although proposals have been made to ground liability in risk, courts have generally held the causation requirement inviolate. This is because causation, as imperfect as are our methods for ascertaining it, particularizes the plaintiff and therefore completes the transaction that calls into play the requirements of corrective justice.⁹⁷

If Dr. Snyder's point, instead, is that the creation of risk is sufficient for the wrongdoing part of the tort (what we call in negligence the breach of duty), I guess my answer is: What's wrong with that? He does not seem to sufficiently appreciate that, especially in toxic exposure cases, the plaintiff has no control over the risk to which he or she is being subjected. Some risk is of course inevitable. But the question then becomes: Why should the plaintiff bear the cost of the materialization of that risk, especially when the defendant is deriving the benefit of the risk's creation, without the plaintiff's input? Recovery for the injury satisfies the hunger of corrective justice, which *is* the special mission of tort law; recovery places the parties in the position they occupied before the defendant's risky conduct caused the plaintiff's injury.⁹⁸

96. Snyder, Medicolegal Controversies, supra note 8, at 373-75; Spivack & Snyder, Breast Implant Cases, supra note 8, at 464-65 n.98.

97. Ernest J. Weinrib, Causation and Wrongdoing, 63 CHI.-KENT L. REV. 407 (1987).

98. As far as workplace injuries are concerned, and assuming the defendant is the employer for present purposes, Dr. Snyder sets a much higher barrier to recovery than I would. Snyder, *Medicolegal Controversies, supra* note 8, at 376-78. Here, I would be quite liberal in deciding that an environmental injury was workplace-related. The parties agree to give up something in the workers' compensation scheme; in exchange for foregoing the right to sue in tort, the plaintiff "buys" a kind of insurance against insolvency when injured on the job. As Dr. Snyder recognizes and as these remarks have shown, the issue of injury has become increasingly difficult when work-related illnesses are included as compensable events. But assuming some demonstration of plausible causal association can be made, the decision to impose liability on the employer seems preferable to forcing the worker to fall back on whatever other insurance she may have, if any. This approach may cause the cost of workers' compensation to increase; however, that increase is better spread across the board than visited on any one worker. Again, the question remains: What is the best we can do under the cloud

^{95.} See, e.g., David Phelps, Document Overload: State Lawyers Use a Flood of Internal Papers to Build Case in Tobacco Lawsuit, STAR TRIBUNE, Nov. 13, 1996, at 1D ("Almost weekly... some new tobacco document hits the public domain and turns up in newspaper headlines, released by plaintiffs' attorneys in an attempt to expose a conspiracy of silence by showing the industry's alleged complicity in hiding the damages of smoking.").

IV. CONCLUSION

In retrospect, my remarks may have come across as more agnostic on causation than I have intended. We can, and do, make serviceable decisions about causation, and toxic torts, while they may be more difficult, do not raise issues that should cause massive hemorrhaging of tort doctrine. The danger is in seeking refuge in a discredited, and now naked, model of causation that offers false certainty. Epidemiologists know all too well the limits of their science; courts can be humble, without being humiliated, in also accepting such recognition. Then, judges can go about the business of deciding the cases brought before them, as they have been doing for centuries.

For pithy epigrammatic statements that can be used to support a host of propositions, there's no one like Gertrude Stein. I'd like to close my comments with one of her less popular quotes:

There ain't no answer. There ain't going to be any answer. There never has been an answer. That's the answer."

99. I read this on a greeting card.